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Modern Concepts of Cardiovascular Disease

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LEFT VENTRICULAR HEART FAILURE

Left ventricular failure is heralded by shortness of breath, as a cardinal symptom. Breathlessness usually appears only after exertion but it may come on at rest, particularly during sleep and may awaken the patient from deep sleep. This significant respiratory distress or dyspnea may be transient, periodic or persistent. Though it may be a symptom of emphysema, anemia, aneurysm or other conditions, dyspnea is most commonly the response to pulmonary engorgement which in turn is the result of congestive left ventricular heart failure. Paroxysmal nocturnal orthopnea often appears as the first indication of left ventricular heart failure. There may be merely an exaggeration of a regularly recurring periodic apnea following a regularly increasing dyspnea of Cheyne-Stokes' breathing. Usually it is evidence of even more striking and serious disturbance in the respiratory center.

The failing left heart gives rise to the characteristic symptom in advance of the development of pathognomonic signs. The presence of primary physical conditions, hypertensive arteriolar disease with or without chronic nephritis, aortic valvulitis with regurgitation or stenosis, that are known to put a great load or strain on the left heart, should cause the physician to be on the alert for evidences that suggest the imminence of failure. These conditions may be present for years before the clinical phenomena of left ventricular failure intervene gradually or suddenly. Slight Cheyne-Stokes' breathing at nightfall, alternation of the pulse and blood pressure or gallop rhythm over the apex and decreasing vital capacity are premonitory signs. As left ventricular failure evolves, fine, crepitant rales and dullness usually appear first at the right lung base posteriorly. Eventually there is a disappearance of tactile fremitus, muffling of the breath sounds and egophony over a developing hydrothorax.

Failure eventually practically always involves the whole heart muscle mass but fairly frequently the symptoms and signs of weakness of the left less often of the right ventricle alone are presented. The specific unilateral manifestations may persist for some time but only rarely in absolutely pure form. The delicately balanced circulatory equilibrium that normally exists between the lesser or pulmonary and the greater or general systems must be recognized. A relatively minute lessening of the output per beat from one ventricle results after a few hours in the accumulation of a significant blood volume rise in the system that is normally drained by the ventricle.

Forward heart failure develops at times under less common circumstances, the result of loss of left ventricular propulsive power as after a massive myocardial infarction. This creates a clinical picture of shock or peripheral circulatory collapse. Backward heart failure is the common type and is of a different nature. Much convincing argument has been set forth in support of the theory that the usual type of circulatory failure is due to back pressure of blood in the incompletely drained venous system. This will explain most of the clinical phenomena, the symptoms and the signs. The two types, backward and forward failure, may be combined, as in some cases of hypertensive heart disease with coronary occlusion, with myocardial infarction and weakness.

Recognition of the mechanism of circulatory failure and of the ventricle that is predominately weakened is requisite to the most rational scientific and logical treatment of a given situation. The characteristic respiratory symptoms of dyspnea, orthopnea, cough, and sometimes frothy expectoration with signs of pulmonary congestion, hypertension and P_2 accentuation, warrant the diagnosis of left ventricular weakness, insufficiency or failure.

General non-specific symptoms of a failing circulation as the sense of exhaustion, indigestion, etc., will not be gone into, nor will the symptoms and signs of the more commonly easily recognized congestive or right heart failure be considered.

The Clinical Picture in Terms of Pathological Physiology. Sudden air hunger may be precipitately presented without any premonitory symptoms. The patient may be awakened from a deep sleep with a start in a state of highly nervous excitation. The attack often causes him to get out of bed and go to a window to "get fresh air." Usually the orthopnea subsides rapidly as the patient gets thoroughly awakened. After a variable interval, often short, the patient is able to go back to sleep and may sleep the rest of the night undisturbed.

There is considerable evidence that the sensitivity of the central respiratory center changes and that showers of nerve impulses from the lung are of most serious moment in the pathological physiology of left ventricular heart failure. The dramatic relief that follows the use of morphine substantiates this. The chronic or acute pulmonary venous stasis distends the lung and pleura which gives rise to stimulation of the respiratory center, the so-called Hering-Breuer reflex. The reflexes carried to the medulla through the vagi, bombard and excite the respiratory center to overactivity.

Great anxiety is the predominant symptom and air hunger seems to make the patient "panicky." The breath seems to go so far and no further. It may be quite short. The respiration is rather shallow and rapid and seemingly ineffectual. Then again there may develop a gasping, sighing, thrashing around, noisiness and restlessness. Shallow, rapid breathing may result from the effect of severe anoxemia on the respiratory center and creates a vicious circle. It dissipates carbon dioxide and reduces the tidal air 50 per cent though the ineffectual dead space may be adequately ventilated. The vital capacity is reduced by the encroachment upon the air sacs of pulmonary congestion.

Paroxysmal nocturnal dyspnea attacks are dramatic episodes of acute left ventricular failure. The explanation of this phenomenon upon the basis of pathological physiology is still quite theoretical but should nevertheless be set forth. There is known to be an accumulation of various acid metabolites in the tissues of the brain stem. Pulmonary congestion leads to the decrease in carbon-dioxide accumulation. Sleep removes the control of the respiratory center from the higher centers and local respiratory center acidosis develops. Carbon-dioxide tension rises and otherwise overactive autonomic stress or vagotonia increases during sleep. The blood pressure and pulse rate usually fall so that the irrigation of the respiratory center as well as the general musculature becomes less adequate. These conditions develop simultaneously to a certain point at which an attack or paroxysm is precipitated, usually during deepest sleep. The systolic and diastolic blood pressure and pulse rate rise sharply as the dyspnea increases.

Acute pulmonary edema of acute left ventricular failure, with the transudation of great supplies of fluid into the bronchioles, sharply reduces the available respiratory surface for gaseous exchange. Increased respiratory distress, cough, cyanosis, frothy pink-stained sputum and the crepitant rales of acute pulmonary edema rapidly develop. The patient may drown in his own extruded pulmonary fluid.

Treatment of the Pathologic Physiology. The success of some emergency therapeutic procedures throw some light on the mechanisms at play in acute left ventricular failure. In profuse transudation *tracheal bronchial catheterization with suction* may be necessary for removal of the secretions and prevention of fatal anoxic anoxia in the patient. The appearance of cyanosis in such a patient is an indication for *prompt administration of oxygen* by means of a noronasal mask in as nearly 100 per cent atmosphere as possible in order to get arterial blood oxygen saturation by mass action. Congestion of the lungs and pulmonary edema interfere with normal arterial oxygen saturation of the blood.

Increased *oxygen* tension in the atmosphere supplied raises that in the alveolar air and results in a nearly normal saturation. Under standard condition, each 100 cc. of blood normally saturated with oxygen carries 18.5 to 20.0 cc. of oxygen. Most of this is in loose combination with hemoglobin and only 0.36 cc. of oxygen is in solution in the plasma. An atmosphere of 100 per cent, raises the level in 100 cc. of oxygen saturated blood to 22 or 23 cc. in the hemoglobin while that carried in physical solution in plasma is raised to 2.2 cc. These figures represent the maximum supersaturation.

The purely mechanical factor of engorgement seems to play a role in that trapping venous blood in the periphery by *tourniquets on all four extremities* usually stops an attack. This maneuver prevents the return of blood to the right side of the heart and consequently limits the output of the right ventricle. This would tend to lower the congestion in the lung. *Phlebotomy* or *venesection* acts similarly. The decreased filling of the right heart causes a drop in the right ventricular output and the imbalance between the right and left ventricular outputs disappears and the equilibrium in the two circulatory systems tends to be reestablished. If both ventricular outputs are diminished equally, the circulation is slowed but general congestive heart failure is avoided.

Other Emergency Therapeutic Measures. The absolute specificity of *morphine sulphate,* (¼ gr. or 15 mg.) subcutaneously, in this condition is the most convincing evidence of central nervous origin of most of the trouble. Anxiety is allayed, excessive reflexes from the lungs are interrupted, the cough stops and sweating and pallor disappear. Cheyne-Stokes' breathing may be precipitated and aggravated. The blood pressures, both systolic and diastolic, drop back to normal. The increased parasympathetic tonus may be relieved by atropine and the cardiac asthma gradually subsides.

Pure or crystalline digitalis glucosides are indicated intravenously as powerful left ventricular heart muscle tonics in all severe attacks of pulmonary edema. Prompt myocardial tonic stimulation is, in my experience, best and most safely accomplished with pure crystalline glucosides, as Digilanid C or Lanatoside C 1½ to 2 mg. I. V., or strophanthin ½ mgm. I. V. It should be remembered that the latter should not be used if the patient has taken digitalis by mouth within two weeks. I have had no serious reactions following any of a considerable number of injections of the newer Digilanid C introduced intravenously in 1½ to 2 mg. doses.

Digitalization with the oral standard powdered leaf of digitalis by the rapid or moderately rapid methods of 4½ gr. (0.3 G) or 3 gr. (0.2 G) for 3 or 5 doses at six-hour intervals usually has a very definite effect in increasing the left ventricular tone and relieving the patient completely of attacks. Immediate support of the left ventricular myocardium by intravenous preparations of digitalis may, however, be necessary to save the situation.

Aminophyllin, intravenously in a 7½ gr. (0.5 G) dose may theoretically be dangerous during the acute phase because of increasing the blood volume which would thus add sharply to the load of the left ventricle. Practically, however, aminophyllin has been used without being followed by any recognized instances of aggravation. It has always seemed rational to me to administer aminophyllin in 3 gr. (0.2 G) doses T.I.D., P.C., for the dilating effects of the drug on the coronary arterial bed. This probable coronary dilating effect is the rational basis for the administration of aminophyllin as a possible method of preventing the onset of attacks of acute left ventricular failure.

Intravenous *mercurial* diuretics and particularly combinations of organic mercurials and xanthines as mercupurin often are effective in interrupting serious regularly recurring nocturnal paroxysms of respiratory distress even in the absence of signs of pulmonary transudation and edema.

Occasionally adjustments take place in the circulation spontaneously and the imbalance in left and right ventricular outputs is naturally overcome. Sometimes at the height of left ventricular failure with extreme pulmonary engorgement, cyanosis and orthopnea, the "safety valve" or the tricuspid ring may dilate and permit regurgitation. As the right ventricle fails the acute and extreme pulmonary engorgement sometimes seems to be relieved, the forcing of the blood from the right ventricle into the congested lung is decreased, the attacks may thus be suspended even though the circulatory failure is not lessened. The shift seems to relieve the situation. The distress is relieved, the respiratory function improves, the breathing is easier, the cyanosis diminishes, the vital capacity rises and the lung fields clear. Accompanying these changes there is engorgement behind the right heart, the liver enlarges and may accept and hold a liter and a half of blood and edema gradually develops. The new problems of general or systematic congestive or right ventricular failure arise for consideration.

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